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Radiology

X-ray examination of the chest was performed in six of the children and aided the diagnosis in two cases. A skull X-ray was performed in only one instance and was not helpful. Negative radiological findings should not deter the clinician from screening relatives in an attempt to confirm the diagnosis of TBM. In our small series, definite contacts were identified by chest X-ray in four out of five cases.

Three of the six children who had CAT scans had been referred initially to the neurosurgical unit to exclude space-occupying lesions. In five of these children the scan showed ventricular dilatation and was, therefore, helpful in diagnosis and management.

Only one child was old enough to have had routine anti-tuberculous screening at school, but, although the Heaf test was positive, the chest X-ray at the time was reported as normal.

Clinical Features

It is traditionally taught that there is a long history of illness in TBM, but some children, particularly younger ones, may have a much shorter history.^{5,8} Convulsions were the presenting feature in up to 20% of children under 2 years in one study,⁵ and one child in our series was admitted with convulsions associated with fever.

Choroidal tubercles were not found in any of the seven cases, which is in agreement with the suggestion⁸ that

choroidal tubercles are found in TBM only in association with miliary tuberculosis; none of the children in our series had miliary tuberculosis.

Conclusions

Although local increases in the frequency of TBM do not necessarily reflect a national trend, we have shown that TBM still occurs in children in the U.K. and it is not confined to immigrant families. The history may be short and convulsions are not uncommon. The use of the wrong tuberculin skin test may delay treatment, and a Mantoux 1:1000 test should be used whenever the diagnosis of TBM is in question. The diagnosis is not excluded by a normal chest X-ray. Contacts should be followed up energetically. A bromide partition test is an additional useful, safe, and easy test.

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REFERENCES

1. Editorial. Tuberculous meningitis in children. *Br Med J* 1971; i: 1-2.
2. Parson MA. Tuberculous meningitis. Oxford: Oxford University Press, 1980.
3. Smith HV, Vollum RL. The diagnosis of tuberculous meningitis. *Br Med Bull* 1954; 10: 140-44.
4. Lunn JA. Reason for variable response to the tine test. *Br Med J* 1980; ii: 223.
5. Lincoln EM, Sordillo SVR, Davies PA. Tuberculous meningitis in children. *J Pediatr* 1960; 57: 807-23.
6. Lorber J. Treatment of tuberculous meningitis. *Br Med J* 1960; i: 1309-12.
7. Taylor LM, Smith HV, Hunter G. The blood-CSF barrier to bromide in the diagnosis of tuberculous meningitis. *Lancet* 1954; ii: 700-02.
8. Illingworth RS. The early diagnosis of tuberculous meningitis. *Br Med J* 1950; i: 479-81.

Public Health

CALICIVIRUS GASTROENTERITIS IN NORTH WEST LONDON

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Summary During a thirty-month study of gastroenteritis in North West London, 592 cases were found to be associated with excretion of viruses. 39 (6.6%) of these patients, most of whom were admitted to hospital because of gastroenteritis, were shedding caliciviruses. The cases occurred throughout the year with a peak incidence in the winter. The 39 patients ranged in age from 6 weeks to 13 years, the peak incidence being among infants aged 1-6 months. The clinical features of calicivirus infection are not distinguishable from those of rotavirus infection.

INTRODUCTION

WITHIN the past decade viruses have been demonstrated by electron microscopy in the stools of those with and those without the symptoms of gastroenteritis. The agents reported include rotavirus, Norwalk-like agents, calicivirus, astrovirus, and coronavirus. The aetiological role of most of these agents is still not clearly defined and further information on the part they play in human diarrhoea has been called for.¹

This report presents some further clinical, serological, and epidemiological data on one of the more recently discovered agents, calicivirus, and is based on a study of 39 sporadic cases in North West London.

MATERIALS AND METHODS

Through the period January, 1979-June, 1981, samples of specimens of stools sent to the laboratory from patients with a history of gastroenteritis or diarrhoea and vomiting were examined by electron microscopy, whether or not such examination was requested. Most of these specimens had been found to be negative for bacterial pathogens (shigella, campylobacter, and salmonella); however, for some of the others we do not know whether pathogenic bacteria were present.

The methods used for faecal examination and for immune electron microscopy (IEM) have been described elsewhere.² Identification was based on demonstration of the characteristic morphological features of the Caliciviridae.^{2,3} When calicivirus particles were detected in a specimen a questionnaire restricted to a small number of features, to be indicated as present or absent, was sent to the clinician with the report. The age and sex of the patient and month when the specimen was collected were obtained from the request forms as these data were regarded as reliable. The information requested was:

1. Date of admission to hospital (if patient was admitted) and date of onset of relevant clinical features.
2. Reason for admission if other than gastroenteritis.
3. Presence or absence of diarrhoea, vomiting, fever, upper respiratory tract infection, and "other features."
4. Duration of features reported.
5. History of "failure to thrive" or malabsorption.
6. Siblings, and if ill or not.

RESULTS

During the period of the study, viruses were detected by electron microscopy in the stools of 592 patients with gastroenteritis. 39 (6.6%) cases were associated with calicivirus excretion. (A separate report on other features of this study is being prepared.) Questionnaires were completed for 28 (72%) of the patients with calicivirus. The data presented are based on these responses except in respect of age and sex of patient and month of infection, which are based on 39 cases.

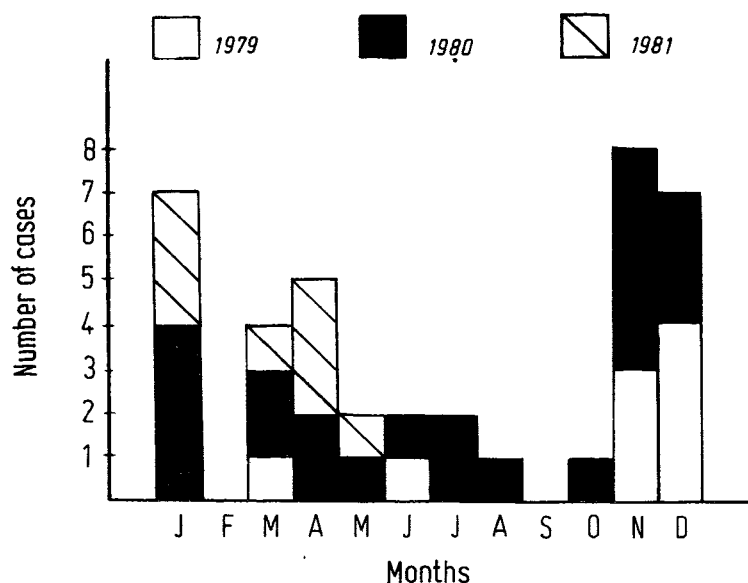


Fig. 1—Monthly distribution of 39 cases of calicivirus infection.

Seasonal distribution.—Sporadic cases occurred throughout the year (fig. 1), with a peak incidence, 22 (56%), in the winter quarter, November to January.

Age.—The age distribution of 37 cases is shown in fig. 2. Two male patients, one aged thirteen years and one of unknown age, are not included. The age-range was six weeks to thirteen years; peak incidence occurred during the first six months of life. No adult infection was detected, although this is known to occur.⁴ Neonatal infection was not encountered, although many stools from babies in special care baby units were included in the gastroenteritis study.

Sex ratio.—The higher infection rate in male children reported for other gastroenteritis viruses was demonstrated among the sporadic cases of calicivirus infection (fig. 2). 24 males were infected and 15 females, a ratio of 1.6/1.

Clinical features.—These were recorded in 28 patients, 26 of whom had been admitted to hospital. Diarrhoea was the commonest clinical feature, occurring in 25 (89%) of the 28 cases. Vomiting was reported in 14 (50%); only 1 patient had vomiting without diarrhoea and this was the only case of projectile vomiting reported. 2 children had neither diarrhoea nor vomiting. Upper respiratory tract infection

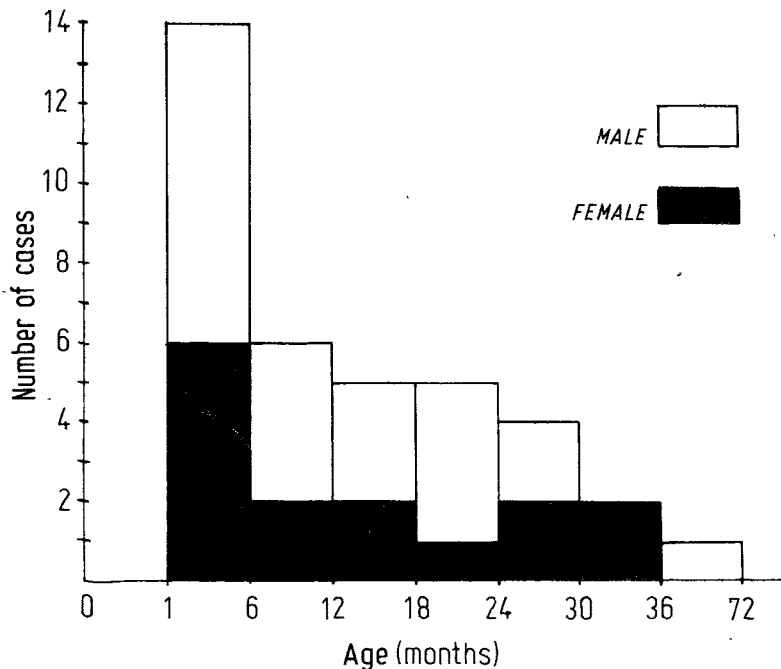


Fig. 2—Age distribution of 37 cases of calicivirus infection.

TABLE I—FAECAL SHEDDING OF VIRUS FOR 16 CASES IN LAST SPECIMEN SAMPLED

Day	EM +ve
1-4	8
5-9	6
10-11	2

EM = electron microscopy.

TABLE II—SEROLOGICAL RELATIONS BETWEEN 4 SPORADIC AND 3 OUTBREAK STRAINS OF CALICIVIRUS

Virus	Serum				
	Portsmouth ⁴		Sapporo ⁵		Roth ²
	Acute	Conva-lescent	Acute	Conva-lescent	Conva-lescent
Portsmouth	<20	320	20/40	20	10/20
Sapporo	20/40	20	<10	320	NA
Roth	NA	NA	NA	NA	512
Piper	<20	20	<10	10/20	160
Oxford	<20	20	<20	<20	≥80
Harrow	<20	<20	<20	<20	≥80
Isleworth	20	40	<20	<20	≥80

was reported in a third of the cases.⁹ Failure to thrive and malabsorption was reported in only 3 cases. 5 children had fever. The duration of symptoms ranged from two to eleven days with most illnesses lasting about four days. 13 (46%) of the affected children had siblings and, of these, 5 had at least 1 sibling with similar symptoms of illness. In 1 case the brother attended a school where there was a concurrent outbreak of diarrhoea and vomiting. The duration of excretion of viruses is shown in table I; the finding that the chances of detecting virus diminishes after the fourth day emphasises the need to obtain specimens during the symptomatic phase of illness.^{2,5}

Serology (immune electron microscopy [IEM]).—Four strains of virus found in sporadic infections were tested against sera from three reported outbreaks^{2,4,6} (table II). There is little evidence of serological relations between the sporadic strains and strains associated with two of the outbreaks, but there is some evidence for a relation with the third.²

DISCUSSION

Viruses morphologically identical with caliciviruses have recently been detected in animals^{7,8} and people⁹⁻¹¹ with gastroenteritis. The study of outbreaks in man in the U.K.,^{2,4,12} Japan,⁶ and Canada¹³ has provided evidence that these viruses can cause diarrhoea and vomiting in all age-groups other than neonates. The apparent absence of cases in neonates may be due to predominantly asymptomatic infection or be associated with immunity due to maternal antibody.

In the series of sporadic cases reported here, diarrhoea was the most frequent symptom, whereas, in children aged four to six years, vomiting was more common.² It has been suggested⁵ that these differences in symptoms may be age-related. However, since vomiting has been reported as the commonest feature in infected infants aged nine days to two years, age does not appear to be the sole determinant.¹³ Other clinical features, such as fever and upper respiratory tract infection, have been reported infrequently. Thus the clinical features of calicivirus gastroenteritis are not distinguishable from those due to rotavirus infection.

Several cases of calicivirus infection associated with failure to thrive have led us to consider the possibility of an

aetiological relation. However, the results of this small survey do not provide much support for this view.

Diarrhoea developed ten or more days after admission to hospital in 3 out of 26 infants, indicating that caliciviruses, like rotaviruses, can be a cause of nosocomial infection.

Little is known about the range of human calicivirus serotypes. Results from this and a previous study⁴ provide evidence for at least three distinct serotypes which have been associated with outbreaks. The limited data obtained on the sporadic cases from North West London suggest that they may be associated with strains from one of these outbreaks.² Whether strain differences, as reflected by IEM serology, have any implications for susceptibility to repeated infection with calicivirus or are, in any way, related to geographical distribution, remains to be established.

Caliciviruses have been identified as a cause of gastroenteritis only in countries where electron microscopy facilities are readily available. Surveys of viral gastroenteritis in these areas indicate that calicivirus accounts for a proportion of gastroenteritis cases (Japan 2%,¹⁰ Scandinavia 5%,¹⁴ and U.K [present study] 6.6%). Caliciviruses have not yet been reported from Africa, India, or Central and South America, where viral diarrhoea (rotavirus) has recently proved to be a major cause of serious morbidity and mortality.

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REFERENCES

1. Report of the Sub-group of the Scientific Working Group on Epidemiology and Etiology: Rotavirus and other viral diarrhoeas (1979). WHO/DDC/EPE/79.2.
2. Cubitt WD, McSwiggan DA, Moore W. Winter vomiting disease caused by calicivirus. *J Clin Path* 1979; **32**: 786-93.
3. Burroughs JN, Doel TR, Smale CJ, Brown F. A model for vesicular exanthema virus. The prototype of the calicivirus group. *J Gen Virol* 1978; **40**: 161-74.
4. Cubitt WD, Paed PJ, Saeed AA. A new serotype of calicivirus associated with an outbreak of gastroenteritis in a residential home for the elderly. *J Clin Path* 1981; **34**: 924-26.
5. Chiba S, Sakuma Y, Kogasaka R, et al. Faecal shedding of virus in relation to the days of illness in infantile gastroenteritis due to calicivirus. *J Inf Dis* 1980; **142**: 247-49.
6. Chiba S, Sakuma Y, Kogasaka R, et al. An outbreak of gastroenteritis associated with calicivirus in an infant home. *J Med Virol* 1979; **4**: 249-54.
7. Woods GN, Bridger JC. Isolation of small viruses resembling astroviruses and caliciviruses from acute enteritis in calves. *J Med Microbiol* 1978; **11**: 441-51.
8. Saif LJ, Bohl EM, Theil KW, Cross RF, House JA. Rotavirus-like, calicivirus-like, and 23 nm virus-like particles associated with diarrhoea in young pigs. *J Clin Microbiol* 1980; **12**: 105-11.
9. Scott TM, Madeley CR, Cosgrove BP, Stanfield JP. Stool viruses in babies in Glasgow. *J Hyg Camb* 1979; **83**: 469-85.
10. Flewett TH, Davies H. Caliciviruses in man. *Lancet* 1976; **i**: 311.
11. Suzuki H, Konno T, Kutsuzawa T, et al. The occurrence of calicivirus in infants with acute gastroenteritis. *J Med Virol* 1979; **4**: 321-26.
12. Cubitt WD, McSwiggan DA, Arstall S. An outbreak of calicivirus infection in a mother and baby unit. *J Clin Path* 1980; **33**: 1095-98.
13. Spratt HC, Marks MI, Gomersall M, Gill P, Pai CH. Nosocomial infantile gastroenteritis associated with minirovirus and calicivirus. *J Pediatr* 1978; **93**: 922-26.
14. Kjeldsberg E. Small spherical viruses in faeces from gastroenteritis patients. *Acta Pathol Microbiol Scand* 1977; **85**: 351-54.

Round the World

From our Correspondents

THE PHYSICIAN IN PAPUA NEW GUINEA

OPPORTUNITIES for specialist physicians wishing to work in tropical developing countries have diminished in recent years. It is still possible to obtain a post in Papua New Guinea, although the positions available now will be filled within the next few years by local graduates. Surprisingly little is known in Europe about the country,* and it is difficult to obtain accurate information about existing work. The work of junior medical officers in Papua New Guinea has, however, been the topic of several recent articles.^{1,2}

The Country

The central highlands are mostly above 5000 feet, and although just south of the equator, the climate is mild with cool nights. As a result, malaria is not prevalent, and the valleys are densely populated; deforestation is accordingly extensive. The coastal regions of the mainland, however, and the large islands to the north and east (Manus, New Ireland, New Britain, and Bougainville) are hot and humid throughout the year; malaria is endemic and population densities are relatively low. The Highlands Highway, which runs from Lae on the coast to Mount Hagen and beyond, is not completely sealed, and poor driving standards together with alcohol abuse contribute to a high rate of motoring accidents.³ In 1979, P.N.G. had 67 deaths per 10 000 registered vehicles.⁴ Communication between Port Moresby and the rest of the country and the outlying islands is by air or sea, although travel by air is expensive.

* P.N.G. is 178 260 square miles in area, and the population is estimated to be 3 168 700

1. Burrows M. Papua New Guinea. *Br Med J* 1977; **ii**: 1465-66.
2. Fairclough JA, Spencer SA. Broaden your mind, narrow your chances? Reflections on short-term medical work in the developing world. *Br Med J* 1981; **i**: 1454-55.
3. Shepherd A. Road traffic accidents: a view from the Highlands. *Papua New Guinea Med J* 1980; **23**: 57-58.
4. Bouraga P. Road traffic accidents and the role of the police. *Papua New Guinea Med J* 1980; **23**: 59.

The Health Services

The health services of P.N.G. are comprehensive and well organised. The Health Department has base hospitals at Port Moresby, Lae, Goroka, Madang, and Rabaul. Port Moresby General Hospital has four general medical units, two of which are directed by university physicians, while the other base hospitals have one physician, surgeon, paediatrician, and obstetrician. Anaesthetics are usually administered by specially trained nurses or resident medical officers. Within the region served by a base hospital are a number of district hospitals staffed by general medical officers (often expatriate) and nurses. The district hospitals in turn provide a referral centre for the health centres in outlying areas, which are manned by health extension officers (H.E.O.s), paramedical workers who have completed a 3-year course in basic medicine. The health centres supervise a number of aid posts which are in the charge of aid post orderlies (A.P.O.s), competent to treat minor maladies and common illnesses such as malaria. This system provides basic medical care for every person, even in remote areas.

The University of Papua New Guinea is situated in Port Moresby. There are three posts for consultant physicians in clinical medicine, one chair and two senior lectureships, one of which is at Goroka. The Papua New Guinea Institute of Medical Research is

